1. **AMENDMENTS TO THE SPECIFICATION:**

***Please replace the paragraph beginning on page 26 at line 16, bridging to line 9 on page 27 with the following amended paragraph.***

Elafin (neutrophil elastase inhibitor) was originally isolated from the scales of patients with psoriasis (Wiedow *et al.*, 1990) and in lung secretions (Sallenave and Ryle, 1991; Tremblay et al., 1996), but it is also present at mucosal sites in many tissues. It presents in sputum, in tracheal biopsies and bronchoalveolar lavage from both normal subjects and patients, and its synthesis by Clara cells and type IT cells in lung. It has recently been observed that macrophages also express elafin. Elafin is a 6-kDa peptide. The sequence of the gene showed that it is approximately 2.3-kb long, and is composed of three exons and two introns. The 5' regulatory sequences contain activator protein-1 and nuclear factor-B sites. A positive regulatory cis-element present in the region between -505 and -368 bp is responsible for the upregulation of the elafin gene in normal breast epithelial cells. The peptide is composed of 1 17 amino acid residues including a hydrophobic signal peptide of 22 residues. Elafin can be divided into two domains, the carboxy-terminal domain containing the antiproteinase active site and the amino-terminal domain containing characteristic VKGQ sequences (amino acids 47-50 of SEQ ID NO:4). These sequences allow the elafin molecule to glue itself into polymers and bind other interstitial molecules through transglutamination. This feature could make elafin maximally effective as a tissue-bound inhibitor as opposed to AAT, which is present in large amounts in the circulation. Elafin has also been suggested to have a locally protective role against neutrophilic damage, presumably because of its small size and negative charge. Elafin has been shown to be more specific in its spectrum. It inhibits pancreatic elastase, neutrophil elastase and proteinase-3.